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A BIOMECHANICAL SIMULATION OF THE EFFECT OF THE EXTRINSIC FLEXOR MUSCLES ON FINGER JOINT FLEXION

D. G. Kamper[1,2], W. Z. Rymer[1,2]
[1]Sensory Motor Performance Program, Rehabilitation Institute of Chicago
[2]Department of Physical Medicine and Rehabilitation, Northwestern University, Chicago, IL, USA

Abstract-**The mechanisms responsible for producing flexion of the metacarpophalangeal (MCP) joints of the fingers remain a matter of debate. Especially, the role of the extrinsic finger flexor muscles has been questioned. Using computer simulation techniques, we sought to determine if it is possible for the extrinsic flexor muscles to produce rotation at all three finger joints and to initiate rotation at the MCP joint. A planar open-link chain with three revolute joints and four links was created to model the index finger. The tendons from the extrinsic flexor muscles were simulated as ropes passing through pulleys. Passive joint stiffness and damping values were obtained from system identification experiments involving the input of angular perturbations to the joint of interest and the measurement of the resulting resistance torque. Simulation output revealed that in the absence of passive joint stiffness and damping, shortening of the extrinsic flexor tendons actually resulted in slight extension of the MCP joint. However, with the inclusion of stiffness and damping, tendon shortening produced simultaneous rotation of all three joints. These results suggest that the extrinsic flexor muscles may play a primary role in controlling MCP flexion[1].**

***Keywords* - index finger, dynamics, stiffness, damping**

I. INTRODUCTION

Finger biomechanics have proven difficult to analyze, due to the seeming redundancy in muscle actuation of the joints, and the potential for each finger muscle to affect multiple joints. For example, the tendons for the extrinsic flexor muscles, the flexor digitorum profundus (FDP) and the flexor digitorum superficialis (FDS) both cross the metacarpophalangeal (MCP) as well as the proximal interphalangeal (PIP) joints. The FDP tendon additionally crosses the distal interphalangeal (DIP) joint, before attaching to the distal phalanx.

While the FDP and FDS tendons cross the MCP joint, they do not attach to the proximal phalanx of the finger or to the metacarpal bone. This has led some to surmise that the FDS and FDP do not contribute to MCP flexion. Others have suggested that FDS and FDP do not initiate MCP flexion. Rather, they form a moment couple about the MCP with the extrinsic extensors through the intrinsic hand muscles [1]. Yet, we felt that the presence of the annular and cruciform pulleys through which the extrinsic flexor tendons pass, coupled with the passive resistance of the PIP and DIP joints would make it possible for FDS and FDP to initiate MCP flexion.

We tested the feasibility of this hypothesis through the creation of a planar computer model of the index finger. While other models of the finger have been developed [2],[3], these models do not incorporate the human passive joint characteristics or physiological pulleys. Passive joint stiffness and damping functions were formulated from experimental data.

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II. METHODOLOGY

A. Computer Simulation

A two-dimensional dynamic model of the index finger was developed using the simulation software Working Model® (Knowledge Revolution, San Mateo, CA). The index finger was used as a representative case, as it is functionally important and also has the greatest independence of all the fingers [4].

The finger was modeled in the mid-sagittal plane, as an open-linked chain with four segments and three degrees-of-freedom. Revolute pin joints were used for the DIP, PIP, and MCP joints. The metacarpal bone was fixed in place (Fig. 1). Phalanx and joint size were obtained from the literature [5]. Segment mass was estimated from segment volume using a constant density (1.1g/cm^3 [6]).

FDS and FDP tendons were modeled as ropes passing through a series of pulleys located on the palmar side of each of the segments. These pulleys represented the anatomical annular and cruciform pulleys (Fig. 1).

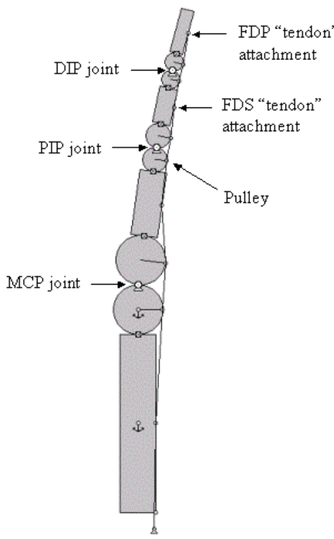

Fig. 1. Model of the index finger with 3 joints and 4 segments. FDP and FDS tendons are represented as ropes that run through pulleys.

Passive joint stiffness and damping were instituted through the use of torque actuators at each joint. Stiffness and damping coefficients were estimated from experimental results.

The nonlinear simulation was run in Working Model® with the FDS and FDP lengths driving the simulation. As a first approximation, these lengths decreased linearly with time. Joint states were updated in the software through

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Abstract

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solution of the dynamics equations using a 4^{th}-order Runge-Kutta algorithm and a 25 ms time step. Angular rotations and velocities were recorded as a function of time.

B. Experimentation

Passive resistance to flexion/extension movement of each of the joints was determined experimentally in human subjects by actuating each joint individually and measuring the resulting resistance torque.

Six healthy adults participated in the experiment. The left index finger was used for each subject. The wrist was placed in fiberglass cast that was subsequently clamped to a table, in order to maintain wrist position and orientation. The index finger was included in this cast up to the middle of the middle phalanx, thereby leaving only the DIP free to flex. The finger was coupled to a servomotor (PMI Motion Technologies, 1.4 HP) through the use of a hose clamp, attached to the servomotor shaft through an aluminum housing. The DIP joint was aligned with the shaft of the servomotor.

Perturbations in joint angle were applied to the finger through the servomotor. Specifically, pseudo-random binary sequences (PRBS) of ±2º in amplitude were imposed at the DIP joint at different operating points [7]. These operating points were spaced 10º apart, ranging from 10º of extension to 60º of flexion. The PID controller of the servomotor was tuned such that the PRBS command signals were low-pass filtered in order to reduce noise. The pass band of the input went beyond 20 Hz, which was considered adequate for the biological parameters of interest.

Joint position, velocity, and torque were measured using an encoder (PMI Motion Technologies, #138647), tachometer (PMI Motion Technologies), and torque meter (Transducer Techniques, TRT-200), respectively (Fig. 2). Data were lowpass filtered at 25 Hz with a 30^{th}-order FIR filter. These resulting data were used to fit an I-B-K model with constant coefficients at each operating point such that

$$\tau = I\ddot{\theta} + B\dot{\theta} + K\theta \qquad (1)$$

The angular acceleration ($\ddot{\theta}$) was numerically computed from the angular velocity using a five-point formula. The I-B-K coefficients were then fit to the data using multiple regression.

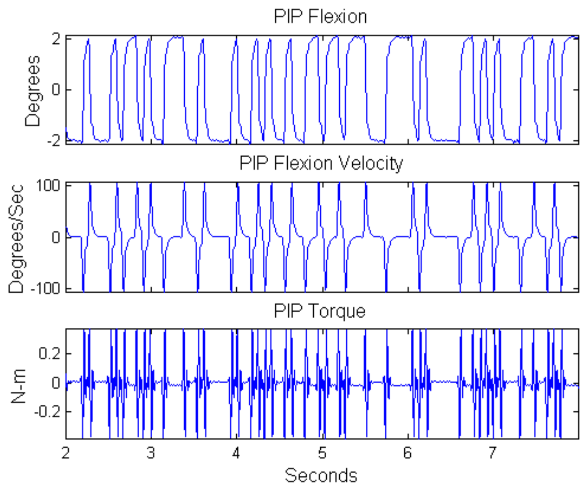

Fig. 2. Example of recorded angular position and velocity and torque.

The cast was then cut back to allow flexion and extension of the PIP joint while the DIP and MCP joints were fixed. Angle PRBS perturbations were applied to the PIP joint and stiffness and damping parameters fit to the data.

Finally, the cast was retracted to the mid palm to expose the MCP joint. Perturbations were again repeated.

For the purposes of the simulation, representative stiffness and damping terms were computed for each joint. The stiffness and damping coefficients were pooled for all of the subjects and polynomials were fit to the data as a function of joint angle through least-squares techniques. The representative joint damping coefficient for each joint was described by a single constant term, independent of joint angle. The representative joint stiffness coefficient was described by a second-order polynomial, relating the stiffness coefficient to the joint angle.

These overall stiffness and damping coefficient equations were then inserted back into the model through the torque actuators, such that

$$\tau_j = B\dot{\theta} + K(\theta - \theta_0) \qquad (2)$$

where τ_j is the passive joint torque and θ_0 is the mean joint angle across the subjects at which the passive torque equals zero. For the DIP joint, θ_0 was set to be a variable, the PIP angle, such that stiffness was a function of the difference between the DIP and PIP angles to account for the action of the extensor hood.

III. Results

A. Passive Stiffness and Damping

For the determination of the passive stiffness and damping, the I-B-K model provided a good fit to the data. All three coefficients were significant ($p < .001$), and the model fit the data well ($R^2 > 0.96$ for each trial).

Joint damping exhibited no ostensible relation with joint angle for any of the three joints (Fig. 3). Regression analyses relating the two were not significant. Thus, joint damping was described by a constant. Joint stiffness exhibited a parabolic relationship with joint angle (Fig. 4), and was thus described by a second-order equation.

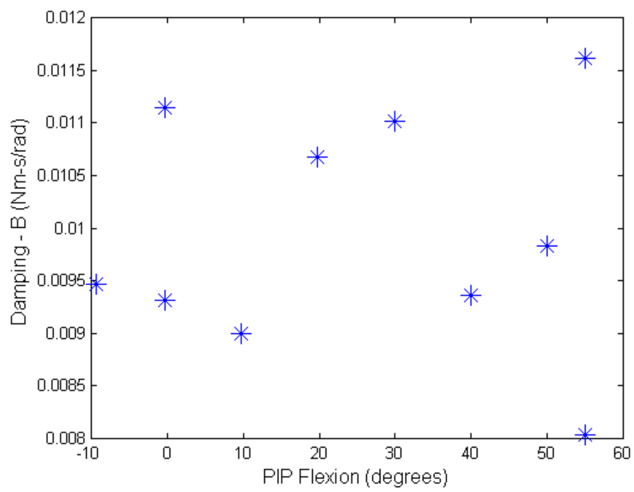

Fig. 3. Sample of PIP joint damping as a function of PIP joint angle for one subject

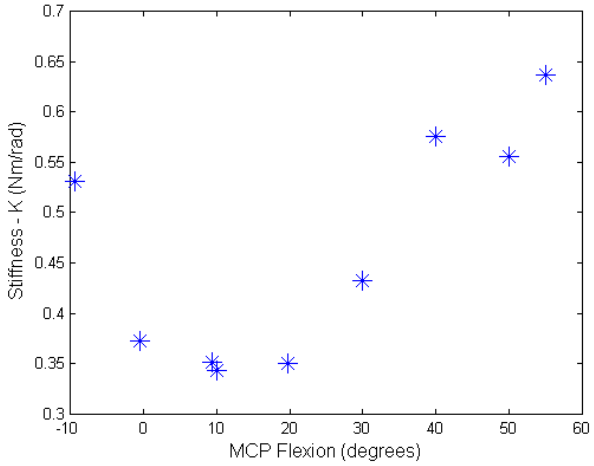

Fig. 4. Sample MCP joint stiffness as a function of MCP joint angle for one subject

Representative stiffness and damping torque equations were then computed from the pooled data (Table I). The data from one subject was not included in the computation of MCP because it exerted undue influence on the data and we were interested in representative data. The nominal stiffness values are similar to those reported in the literature [8]. As expected, both stiffness and damping increased with joint diameter in the distal to proximal direction. Across the range of joint angles, ANOVA results and post-hoc Tukey tests revealed that both the stiffness and damping coefficients were statistically distinct (p<.001) for each joint

TABLE I – REPRESENTATIVE STIFFNESS AND DAMPING PARAMETERS

Joint	Parameters		
	B (N-m-s/rad)	K (N-m/rad)	θ_0 (rad)
DIP	0.0081	$0.384*\theta^2 - 0.089*\theta + 0.133$	θ_{PIP}
PIP	0.0105	$1.058*\theta^2 - 0.760*\theta + 0.396$	0.079
MCP	0.0142	$1.019*\theta^2 - 0.541*\theta + 0.454$	0.275

B. Simulation

The computer simulation was run both with and without the inclusion of the passive stiffness and damping. Without the stiffness and damping, FDP and FDS shortening resulted in slight MCP extension rather than flexion, and DIP extension was greatly limited (Fig. 5).

Inclusion of the physiological passive stiffness and damping in the computer simulation resulted in simultaneous rotation of all three joints in response to FDP and FDS shortening (Fig. 6). For this simulation, joint angles were initially set to their neutral positions at which no passive torque was applied. The mean rate of change for MCP flexion angle over the first 0.3 seconds of movement (0.735), as estimated by the slope of the linear regression, was greater than the rates of change for either PIP (0.598) or DIP flexion (0.472). DIP and PIP flexion increased at much faster rates after this time, and the MCP joint experienced slight extension due to the relatively large passive restoring force.

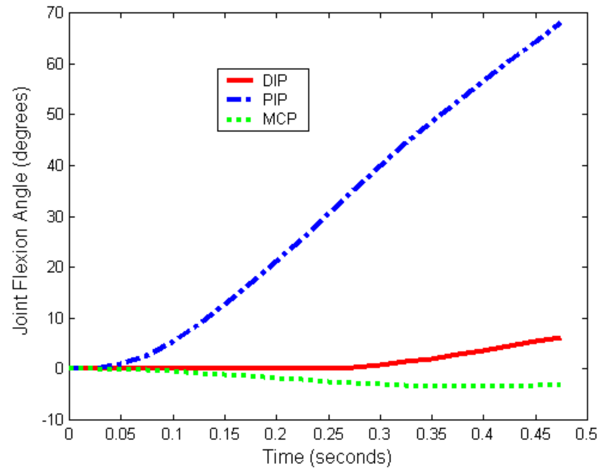

Fig. 5. Joint angles from simulation of FDS and FDP shortening with no joint passive stiffness or damping. DIP: solid line; PIP: dashed-dotted line; MCP: dotted line.

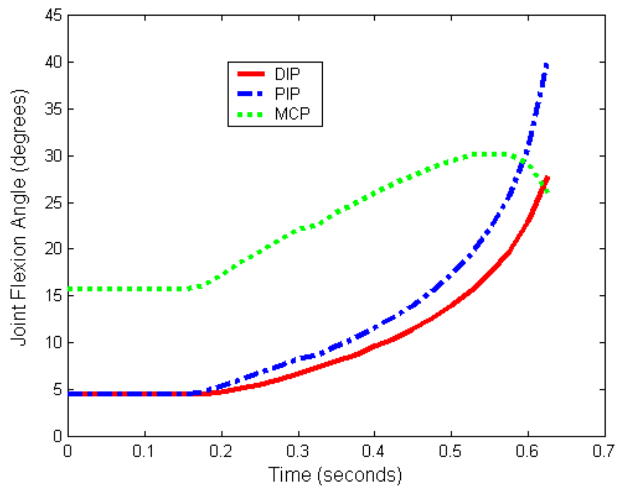

Fig. 6. Joint angles from simulation of FDS and FDP shortening with the inclusion of physiological stiffness and damping.

To test the importance of the pulley mechanisms in producing the simultaneous flexion, we performed a further simulation in which the pulley structures were removed. This resulted in much less rotation of the finger joints, especially PIP and DIP, for the same rate of FDS and FDP shortening (Fig. 7). Mean rates of change over the first 0.3 seconds of movement, during which period the tendons were taut, were 0.462 for MCP, 0.281 for PIP, and 0.236 for DIP. Eventually, the FDP tendon becomes slack while the FDS tendon remains taut.

IV. DISCUSSION

The experimental results revealed that passive finger joint mechanics were well modeled by a linear model with inertial, stiffness, and damping terms. Muscles contribute significantly to the stiffness and damping [9]. Ligaments will also contribute to stiffness as the joint approaches the limit of its range of motion. Synovial fluid surrounding the joint will

add to the viscosity. Finally, skin tissue contributes to the rotational resistance, especially in regard to MCP rotation.

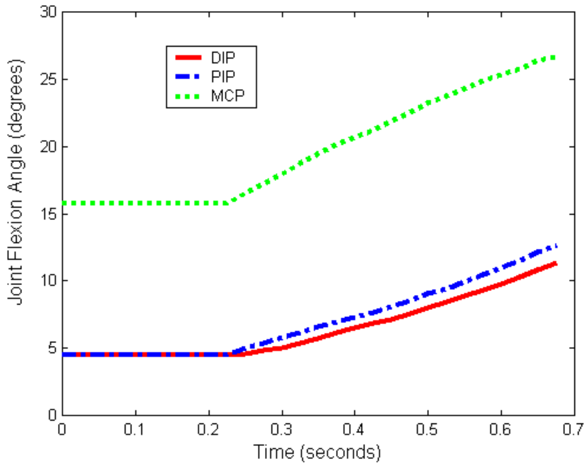

Fig. 7. Joint angles resulting from FDS and FDP shortening with the pulley mechanisms removed (stiffness and damping still included).

Across the DIP, PIP, and MCP joints, stiffness and damping increased with a proximal progression. The increase is partly attributable to the larger diameter of the joint; for an equivalent angular rotation, muscle stretch and stretch velocity of the muscles crossing that joint are greater in the more proximal joints due to the to their larger diameter. Additionally, a greater number of muscles cross the MCP joint than cross the DIP joint. Increases in damping may also be due to the larger amount of fluid in the larger joints that is displaced during rotation.

Joint damping was not related to the nominal joint angle. This may be explained in part by the fact that muscle damping is not dependent on the absolute fiber length and that synovial viscosity is independent of joint angle. Joint stiffness, however, had a strong dependence on joint angle, as might be expected from the nonlinear relationship between absolute fiber length and elastic muscle force [9].

The model simulations suggest the importance of these passive joint characteristics in control of the fingers. Without the inclusion of the passive stiffness and damping, shortening of the simulated FDP and FDS tendons failed to produce flexion of the MCP joint. In fact, the MCP joint was slightly extended even as the PIP joint flexed. Flexion in the DIP joint was very slight.

When the passive stiffness and damping were included, MCP, PIP, and DIP flexion occurred simultaneously. In fact, initial MCP flexion velocity was greater than that for the other two joints. This suggests that FDP and FDS may be able to initiate MCP flexion. This flexion could occur even without the contraction of other finger muscles, such as the intrinsics. Also, no direct connection between the extrinsic flexors and the proximal phalanx, such as the one provided by the lumbrical, is required. Alterations that reduce joint stiffness below normal levels, such as might occur following tendon releases, may actually make finger control more difficult.

Removal of the pulley mechanisms of the finger resulted in considerably less joint flexion for a given amount of shortening of FDS and FDP, especially for the PIP and DIP joints. Absence of the pulleys allowed the tendons to bowstring away from the segments, thereby changing the line of action of the tendons and the effective moment arms. After a certain point, the FDP tendon became slack, while the FDS tendon remained taut. These results suggest that the pulley mechanisms serve both to improve the flexion capabilities of the extrinsic flexors and to maintain tension in the tendons.

V. CONCLUSION

The experimental results indicate that passive joint stiffness and damping are present and significant in the finger joints. These passive characteristics make it theoretically possible for the extrinsic finger flexors to produce simultaneous flexion of all three finger joints, including the metacarpophalangeal joint, without contraction of any other finger muscles. Studies are currently ongoing to compare our simulation results with finger movements produced by electrical stimulation of the flexor digitorum profundus and superficialis.

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